Claims:

Please amend the claims as follows:
Please amend claims 5-8, 12-14, 17-22, 25-27, 30, and 31.
Please add new claims 32-48.

1. **(Original)** A method for treating a disorder in which TNFα activity is detrimental comprising administering to a subject an effective amount of a TNFα inhibitor in a low dose therapy, such that the disorder is treated.

2. **(Original)** The method of claim 1, wherein the disorder is arthritis.

3. **(Original)** The method of claim 2, wherein the disorder is rheumatoid arthritis.

4. **(Previously presented)** The method of claims 2 and 3, wherein symptoms selected from the group consisting of bone erosion, cartilage erosion, inflammation, and vascularity, are treated.

5. **(Currently amended)** The method of any one of claims 1-3 ~~1-4~~, wherein the TNFα inhibitor is an anti-TNFα antibody, or an antigen-binding portion thereof, or a TNFα fusion protein ~~D2E7~~.

6. **(Currently amended)** The method of claims ~~5~~ ~~1-4~~, wherein the TNFα fusion protein ~~inhibitor~~ is etanercept. ~~Etanercept or Remicade~~.

7. **(Currently amended)** The method of claim 32, ~~claims 1-4,~~ wherein D2E7 ~~the TNFα inhibitor~~ is administered in a low dose comprising 0.01 – 0.1 ~~2.0~~ mg/kg.

8. **(Currently amended)** A low dose method to alleviate symptoms associated with a disorder in which TNFα activity is detrimental, comprising administering a low dose of a TNFα inhibitor to a subject suffering from said disorder, such that the symptoms are treated.

9. **(Original)** The method of claim 8, wherein the disorder is arthritis.

10. **(Original)** The method of claim 9, wherein the disorder is rheumatoid arthritis.

11. **(Previously presented)** The method of claims 9 and 10, wherein symptoms are selected from the group consisting of bone erosion, cartilage erosion, inflammation, and vascularity.

12. **(Currently amended)** The method of any one of claims 8-10, ~~claims 8-11,~~ wherein the TNFα inhibitor is an anti-TNFα antibody, or an antigen-binding portion thereof, or a TNFα fusion protein.

13. **(Currently amended)** The method of claim 12, ~~claims 8-11,~~ wherein the TNFα fusion protein ~~inhibitor~~ is etanercept ~~Etanercept or Remicade~~.

14. **(Currently amended)** The method of claim 33, ~~claims 8-13,~~ wherein D2E7 ~~the TNFα inhibitor~~ is administered in a low dose comprising 0.01 – 0.1 ~~2.0~~ mg/kg.

15. **(Original)** A method for treating arthritis comprising administering to a subject an effective amount of a TNFα inhibitor in a low dose therapy, such that the arthritis is treated.

16. **(Original)** The method of claim 15, wherein the arthritis is rheumatoid arthritis.

17. **(Currently amended)** The method of ~~claims~~ either claim 15 or ~~and~~ 16, wherein arthritis is treated by alleviating symptoms selected from the group consisting of bone erosion, cartilage erosion, inflammation, and vascularity.

18 **(Currently amended)** The method of claim 15 or 16 ~~claims 14-17~~ wherein the TNFα inhibitor is an anti-TNFα antibody, or an antigen-binding portion thereof, or a TNFα fusion protein ~~D2E7~~.

19. **(Currently amended)** The method of claim 18 ~~claims 14-17~~ wherein the TNFα fusion protein ~~inhibitor~~ is etanercept ~~Etanercept or Remicade~~.

20. **(Currently amended)** The method of claim 34, ~~claims 15-19,~~ wherein ~~the TNFα inhibitor~~ D2E7 is administered at a low dose comprising 0.01 – 0.1 ~~2.0~~ mg/kg.

21. **(Currently amended)** A low dose method for treating symptoms associated with arthritis comprising administering to a subject a low dose of an effective amount of a TNFα inhibitor, such that the symptoms are alleviated.

22. **(Currently amended)** The method of claim 21, wherein the arthritis is rheumatoid arthritis.

23. **(Previously presented)** The method of claims 21 and 22, wherein the symptoms are selected from the group consisting of bone erosion, cartilage erosion, inflammation, and vascularity.

24. **(Original)** The method of claim 23, wherein the symptoms are further selected from the group consisting of joint distortion, swelling, joint deformation, ankylosis on felxion, and severely impaired movement.

25. **(Currently amended)** The method of claims 21 or 22 ~~claims 21-24~~, wherein the TNFα inhibitor is an anti-TNFα antibody, or an antigen-binding portion thereof, or a TNFα fusion protein ~~D2E7~~.

26. **(Currently amended)** The method of claim 25 ~~claims 21-24~~, wherein the TNFα inhibitor fusion protein ~~inhibitor~~ is etanercept ~~Etanercept or Remicade~~.

27. **(Currently amended)** The method of claim 35, ~~claims 21-26,~~ wherein D2E7 ~~the TNFα inhibitor~~ is administered at a low dose comprising 0.01 – 0.1 ~~2.0~~ mg/kg.

28. **(Original)** A method of sequestering TNFα into complexes in a subject suffering from a disorder in which TNFα activity is detrimental, by administering a low dose of a TNFα inhibitor to the subject.

29. **(Original)** The method of claim 28, wherein the serum level of TNFα is higher than the serum level of TNFα in a subject not suffering from a disorder in which TNFα activity is detrimental.

30. **(Currently amended)** The method of claims ~~28~~ ~~29~~, wherein the anti-TNFα inhibitor is an anti-TNFα antibody, or an antigen-binding portion thereof, or a TNFα fusion protein ~~D2E7~~.

31. **(Currently amended)** The method of any one of claims 1, 8, or 15 ~~1–30~~, wherein the TNFα inhibitor is administered with an additional therapeutic agent.

32. **(New)** The method of claim 5, wherein the anti-TNFα antibody, or an antigen-binding portion thereof, is either infliximab or D2E7.

33. **(New)** The method of claim 12, wherein the anti-TNFα antibody, or an antigen-binding portion thereof, is either infliximab or D2E7.

34. **(New)** The method of claim 18, wherein the anti-TNFα antibody, or an antigen-binding portion thereof, is either infliximab or D2E7.

35. **(New)** The method of claim 25, wherein the anti-TNFα antibody, or an antigen-binding portion thereof, is either infliximab or D2E7.

36. **(New)** The method of claim 30, wherein the anti-TNFα antibody, or an antigen-binding portion thereof, is either infliximab or D2E7.

37. **(New)** The method of claim 30, wherein the TNFα fusion protein is etanercept.

38. **(New)** The method of claim 6, wherein etanercept is administered in a low dose comprising 0.01 - 1.0 mg/kg.

39. **(New)** The method of claim 32, wherein infliximab is administered in a low dose comprising 0.01 – 0.5 mg/kg.

40. **(New)** The method of claim 5, wherein the anti-TNFα antibody, or an antigen-binding portion thereof, is human.

41. **(New)** The method of claim 40, wherein the anti-TNFα antibody, or antigen-binding portion thereof, dissociates from human TNFα with a K_d of 1×10^{-8} M or less and a K_{off} rate constant of 1×10^{-3} s^{-1} or less, both determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1×10^{-7} M or less.

42. **(New)** A low dose method for treating rheumatoid arthritis in which TNFα activity is detrimental comprising administering to a subject a low dose of a

human TNFα antibody, or an antigen-binding portion thereof, such that the disorder is treated.

43. **(New)** The method of claim 36, wherein symptoms selected from the group consisting of bone erosion, cartilage erosion, inflammation, and vascularity, are treated.

44. **(New)** The method of claim 36, wherein the anti-TNFα antibody, or antigen-binding portion thereof, dissociates from human TNFα with a K_d of 1×10^{-8} M or less and a K_{off} rate constant of 1×10^{-3} s^{-1} or less, both determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1×10^{-7} M or less.

45. **(New)** The method of claim 36, wherein the anti-TNFα antibody, or antigen-binding portion thereof, is D2E7.

46. **(New)** The method of claim 36, wherein the amount of antibody, or antigen-binding portion thereof, administered to the subject comprises 0.01 – 0.1 mg/kg.

47. **(New)** The method of any one of claims 1, 8, 15, or 21, wherein the amount of anti-TNFα antibody, or an antigen-binding portion thereof, administered to the subject comprises 0.01 – 0.1 mg/kg.

48. **(New)** A low dose method of improving symptoms in the joints of a subject having arthritis comprising administering to the subject a low dose of a human anti-TNFα antibody, or antigen-binding portion thereof, comprising 0.01-0.1 mg/kg such that at least one symptom selected from the group consisting of inflammation, cartilage erosion, bone erosion, and vascularity is improved.

49. **(New)** The method of claim 44, wherein the anti-TNFα antibody, or antigen-binding portion thereof, is D2E7.